24. *Pathology of Angina Pectoris.*—MM. A. EULENBURG and P. GUTTMAN discuss (*Archiv für Psychiatrie*, t. ii. 1869) all the facts known and the theories advanced in regard to this affection, and they conclude that it is a neurosis both of motion and sensation. The symptoms to which it gives rise may be excited by causes of a different nature, even extraneous to the heart. All the cardiac nerves are probably more or less affected in this disease, and the variation in the phenomena observed in different individuals results doubtless from the more or less active part taken by the nerves which unite together in the cardiac plexus in the production of these phenomena. It is probable that the *great sympathetic* performs the most important part, inasmuch as it constitutes the larger portion of the cardiac plexus.—*Archives Générales*, Sept. 1870.

25. *Intermittent Fever latent for several Months.*—Prof. W. BRAUNE states that of twelve persons who, in the autmn of 1868, had resorted to Borkum, an East Friedland island, for the purpose of taking sea-baths, eleven were attacked with intermittent fever. But while two of the eleven had the fever whilst on the island, the other nine did not suffer from it till the following spring and summer—*i. e.*, from six to nine months after their stay at Borkum. The patients were adults, all of high social position, and most of them females, and belonged to various localities, as Leipzig, Halle, Liegnitz, Atteburg, &c.; and in these places, when the patients severally residing in them were attacked, intermittent fever did not prevail, or was present in only a few sporadic cases. None of the patients had ever before had intermittent fever. It seemed, therefore, that in all of them the malady must have had its cause in malarious poison received during their autumnal stay in Borkum. The summer had been there, as elsewhere, very hot, and there was a considerable scarcity of pure water fit for drinking. The professor remarks that a prolonged period of incubation or latency after malarious poisoning is not so rare as some authorities, as Griesinger, have supposed; and in support of this he appeals to the observations made by Pfeiffer in the army of Weimar, published in a *Jeniaschen Zeitschrift*, 1868. Twice—in 1849 and 1867—certain divisions of the troops suffered an epidemic of intermittent fever, which must have been due in each instance to exposure to malarious poison in the autumn of the preceding year.—*Brit. and For. Med.-Chir. Review*, Oct. 1870, from *Archiv f. Heilkunde*, Dec. 1869.

26. *Vaccinia and Varioloid at Eleven Days' Interval.*—M. BROARDEL communicated to the French Academy of Sciences (22d July, 1870) the following interesting example of such occurence: A woman was admitted into l'Hôpital la Pitié, July 5, 1870, and the next morning at four o'clock was delivered of twins. About noon of the same day both were vaccinated with Jennerian virus from a healthy child, one with nine, the other eight punctures, all of which took; and eight days afterwards (July 13) virus was obtained from them for vaccinating ten or twelve infants. On the 19th of July the twins simultaneously exhibited red spots on their skin which proved to be the commencement of a varioloid eruption. Both children recovered. Both had well-marked vaccine scabs on their arms while suffering from varioloid.—*Archiv. Gén.*, August 30, 1870.

27. *Hemiplegia from Cerebral Hemorrhage in Cases where there is Valvular Disease of the Heart.*—The mere fact that hemiplegia has occurred in a person, even a young person, the subject of valvular disease of the heart, must not alone lead one to the diagnosis of embolism. The mode of onset is the important matter in the diagnosis of the nature of the lesion in all cases of hemiplegia. If we learn that the patient had been deeply insensible for many hours, or, more generally, if he had been for some time in the "apoplectic condition," the diagnosis of cerebral hemorrhage is at least as likely as that of plugging, notwithstanding that there is valvular disease. The bleeding is from an aneurism of the middle cerebral artery, or from a branch of it. It is true that, as a rule, such aneurisms burst outside the brain, and do not produce hemiplegia, but now and then they imitate, so to speak, ordinary cerebral hemorrhage, and break up the motor track. Dr. Hughlings Jackson's observations confirm the opinion of Dr. John W. Ogle and Dr. Church as to the association of aneu-

risms of the larger cerebral arteries with vegetations on the heart's valves. Dr. Hughlings Jackson thinks that the kind of defect of speech which sometimes accompanies hemiplegia is useful evidence as to the *pathological* nature of the lesion. *Loss* of speech—loss excepting the patient's stock word or phrase—is in favour of hemorrhage. Still, plugging will produce complete and permanent loss of speech. If the patient recover, and if during recovery he make frequent mistakes in words—the so-called loss of memory for words—articulating them clearly, Dr. Hughlings Jackson thinks the diagnosis of plugging more warrantable than that of clot.—*British Medical Journal*, Oct. 29, 1870.

28. *Differences in Nervous Symptoms from Differences in the Pathological Nature of Lesions.*—It may seem at first glance that the *pathological* nature of the lesion is of no consequence so far as the kind of symptom goes. Of course, if the very same part of three nervous systems were suddenly destroyed by three different lesions, the resulting symptoms would be the same in the three cases. But practically this does not happen. Although clot, softening from embolism, and tumour, may each damage the region of the corpus striatum, they do not affect the very same parts of this region, and they do not act on it in the very same way. For instance, clot destroys locally, it destroys suddenly, and it squeezes quickly. It mostly destroys more or less of the corpus striatum, and mostly leaves convolutions undamaged, although split off from the motor centre. Softening from plugging occupies arterial districts, and different districts, according to the branch of the Sylvian artery plugged. It may affect convolutions as well as the motor centre. Moreover, it will *alter* the nutrition of some parts while it arrests that of others. After partial or seemingly complete recovery from hemiplegia due to embolism, the patient becomes occasionally liable to chronic convulsive seizures, beginning in the face, or arm, or leg of the side previously paralyzed. This liability does not occur from clot. A tumour may grow anywhere in the district; and, if it grow to a large size very slowly, even in the convolutions near to the corpus striatum, it need produce no symptoms. Nervous organs bear slow squeezing very well indeed; they resent sudden squeezing. The slow growth of tumours may, Dr. HUGHLINGS JACKSON thinks, account for the absence of speech-defects in some cases of disease (especially syphilitic disease) near to the left corpus striatum.

The *altered* nutrition above spoken of is probably owing to blocking of small arteries, which leads to congestion and even to extravasation *beyond* the block. Hence, in cases of embolism of a large branch of the middle cerebral artery, we occasionally find not only softening and destruction of fibres, but also evidences of rupture of small vessels—"red softening" in the convolutions. Sometimes a considerable part of the pia mater is stained of an ochre colour from old extravasation. The expression "altered nutrition" is used because. although "increased nutrition" will result from a locally increased quantity of blood, such congestion will in all probability lead to new matter of Composition, differing from, although of the same Constitution as (using the words in their chemical sense), that of health.—*Ibid.*

29. *Acute Atrophy of the Liver and Phosphorus Poisoning.*—Dr. BOLLINGER describes two cases of phosphorus poisoning and two others of acute yellow atrophy of the liver, according to the results exhibited in their investigation, both macro and microscopically, and gives, thereupon, a summary of these diseases, and especially of the opinions hitherto brought forward of the origin of the so generally concomitant icterus. Contrary to numerous assertions the author notes the absence of the overgrowth of small cells of the interstitial tissue of the liver. In regard to the icterus the author declares for himself that it is in both diseases a *resorption-icterus*, and certainly not, as Virchow has in most cases made it to be received, as being set up by swelling of the mucous membrane at the outlet of the ductus choledochus, but by parenchymatous inflammatory fatty degeneration of the liver cells, and throwing off of the fatty degenerated epithelium of the fine biliary canaliculi. The theory of the hæmatogenous icterus appears very doubtful, as in the liver the effect was